THE ARTIFICIAL HEART-LUNG APPARATUS – EXPERIMENTAL CREATION AND REPAIR OF INTERVENTRICULAR SEPTAL DEFECTS*

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In July of this year, our group, working at the State University of New York College of Medicine at New York City, reported the development of a pump-oxygenator apparatus, and application of it to a patient with intractable cardiac failure.[1] The present report is concerned with further application of this apparatus in the creation and closure of interventricular septal defects in experimental animals. Gibbon *et al.* have described previously creation and repair of interventricular septal defects employing a staged procedure.[2]

METHOD

The essentials of the apparatus are indicated in Figure 1. Blood is withdrawn from the venous system of the subject, passed through a flow meter, and on to slowly revolving stainless steel screen discs, which serve as an oxygenator. It is collected below the discs, passed through a bubble remover consisting of stainless steel sponge coated with Dow-Corning anti-foam, and pumped by a modified Dale-Schuster pump back into the arterial system of the subject. Catheters are inserted into the superior and inferior venae cavae to siphon venous blood to the oxygenator. Sling ligatures are used to occlude the venae cavae adjacent to the heart. The apparatus has the ability spontaneously to film additional screening surfaces if the need for them should develop in the course of perfusion. The pump has an automatic

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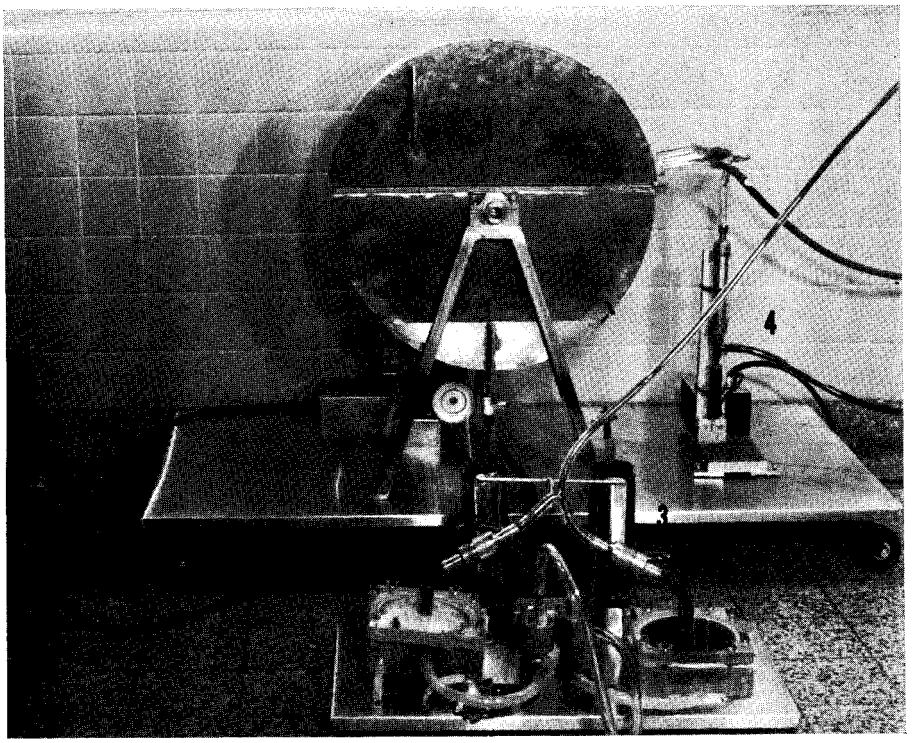

Fig. 1. 1. Oxygenator, 2. Modified Dale-Schuster pump, 3. Bubble trap, 4. Flowmeter.

safety factor in that it injects blood into the arterial system of the subject only if the pump is filled by gravity from the oxygenator prior to each stroke.

The blood utilized to prime the apparatus is prevented from clotting by the addition of 20 mg. of heparin (Connaught Laboratories) for each 500 ml. of freshly drawn arterial blood. The animal to be perfused is given 2.5 mg. of heparin for each kg. of body weight, and the titer is confirmed by protamine titration.

While the right ventricle is open, a Foley catheter is in the right atrium (Fig. 2). Distension of the balloon serves to occlude the tricuspid orifice, and blood returns from the coronary sinus to the oxygenator through the lumen of the catheter. The coronary sinus return in a 15 kg. dog during perfusion is from 40 to 50 ml. per minute.

The ventriculotomy is made longitudinally over the right ventricular outflow tract and is approximately 6 cm. long. The septal defect, usually 2 cm. long, is made in such a position as to avoid the conduction system insofar as possible. The septal defect is closed with 2 to 4 interrupted silk sutures. The ventriculotomy is closed with interrupted or running silk sutures.

At the end of perfusion, a second protamine titration is performed to estimate the amount of protamine needed to return the clotting time to normal. The protamine is given in 5 per cent glucose in water slowly over a period of approximately 15 minutes, following which additional protamine

titrations are performed and additional protamine administered as indicated. The subject is transfused with 200 to 400 ml. of fresh whole blood.

RESULTS

Seventeen consecutive animals were operated upon as described. These dogs weighed from 8 to 33 kg. Two fatalities occurred in these 17 perfusions. The first resulted from a pneumothorax during the postoperative hours when one of the thoracostomy tubes accidentally became disconnected from the suction apparatus. The second fatality followed inadequate care of the drainage tubes, and the animal died with a hemothorax and a bilateral compression atelectasis. In neither instance was the pump-oxygenator felt to be at fault.

Two additional dogs required re-exploration for the control of excessive bleeding from the chest during the postoperative period. In one, the hemorrhage came from the right auricular appendage and in the other from the internal mammary artery. In the remaining dogs, the average postoperative blood loss into the water-seal bottles was 180 ml.

COMMENT

Air embolism has not proved to be a problem in this series. It has been our experience that pumping systems which reduce the pressure of contained blood far below the atmospheric pressure invite bubble formation both from

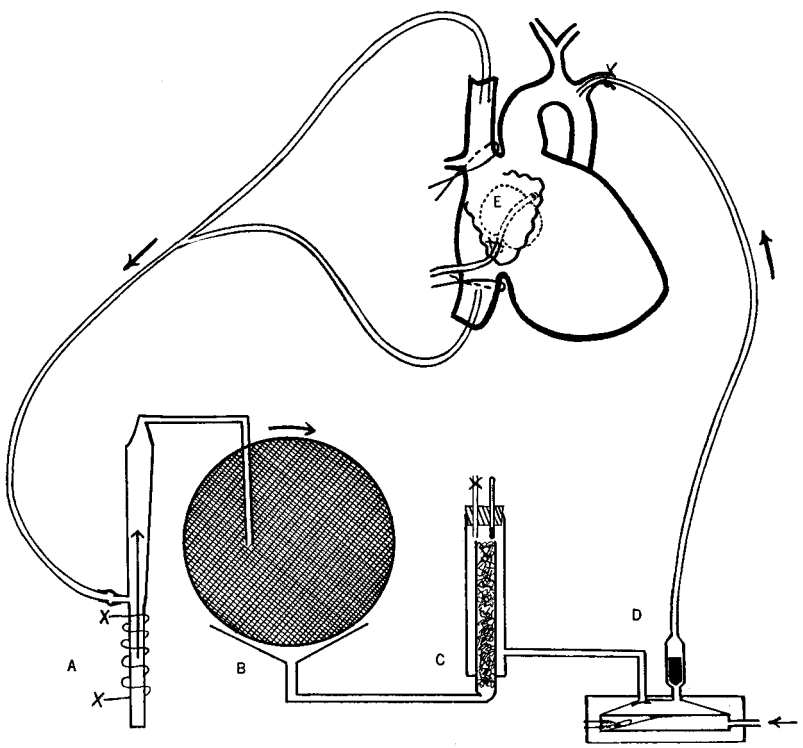

Fig. 2. a. Flowmeter, b. Oxygenator, c. Bubble trap, d. Modified Dale-Schuster pump, compressed air enters at arrow, e. Foley catheter in right atrium.

leakage of atmospheric air through defects in the system which are difficult to prevent and from effervescence of gas previously held in solution. Therefore, we have placed the oxygenator below the level of the subject and used gravity alone to drain the cavae. For the same reason, we have utilized gravity alone to effect filling of the arterial pump from the oxygenator. The self-regulatory nature of the modified Dale-Schuster pump is an additional safety factor. In addition, all connections, tubing, and pump heads are made of transparent materials; this factor facilitates removal of air bubbles from the system prior to perfusion.

Ventricular fibrillation occurred during cardiotomy in 6 of the 17 dogs. We suspect that both temperature changes and injury to the conduction system may be factors. In every instance, defibrillation was easily accomplished with 1 or 2 electrical discharges of 0.1 second at 1.75 amperes. Defibrillation is undoubtedly facilitated by uninterrupted perfusion of the myocardium with well-oxygenated blood. In those hearts which were defibrillated, it was observed that initially a 3:1 atrioventricular block developed, which shortly changed to a 2:1 block. Within a few minutes, this was replaced by a normal sinus rhythm.

The ultimate fate of the ventriculotomy wounds has been a source of some concern. These wounds have been observed for periods up to 5 months. No aneurysm has been present upon sacrifice of any animal. A group of dogs is being observed for longer periods after operation.

CONCLUSION

The pump-oxygenator apparatus which this group has previously reported has been utilized in a series of animal experiments in which ventricular septal defects have been created and closed. There appears to have been no mortality attributable to the utilization of the apparatus.

This apparatus has thus far also been utilized on 4 patients. In 2, the patients have been distinctly benefited, 1 with apparent permanent cure. In this overall experience, there seems to us to be every cause for optimism, and we now seek further appropriate clinical material.

This apparatus has been employed in 4 clinical cases to date. The first was reported by Newman et al. in July of 1955, in which it was possible with a 4 hour perfusion to relieve intractable cardiac failure which had proved resistent to other means of management. The second case was one in which an interventricular septal defect was present. The amount of back bleeding from the aorta was immense, and it is the consensus of the group that so much blood was lost from this source that inadequate peripheral blood pressure was maintained permitting air to enter through the relaxed aortic valve leaflets. Air embolism occurred following the addition of sufficient blood to restore the peripheral blood pressure. The third patient was one in which a pulmonic infundibular stenosis with atrial septal defect had been diagnosed. The septal defect was readily closed through an atriotomy incision. The ventricular incision revealed no evidence of pulmonic stenosis. The patient has made an uneventful recovery and appears to have been cured. The fourth case is an additional patient with interatrial septal defect and rather advanced age, 29 years, in whom exploration revealed a totally unsuspected post-rheumatic pericarditis with dense adhesions, a persistent atrioventricular canal, and, in addition, rheumatic mitral insufficiency. The defect was closed successfully without development of myocardial irregularities, but the patient died of low output right cardiac failure approximately 10 hours after the completion of the procedure.

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